AMENDMENTS TO THE CLAIMS

17. An isolated estrogen receptor-β comprising the sequence depicted in Figure 4, SEQ ID. NO:2.

18. An isolated estrogen receptor-β comprising amino acids 1-45 of the sequence depicted in Figure 4 SEQ ID NO:2.

19. A method for identifying hERβ-interactive compounds, said method comprising:

(a) contacting the polypeptide of claim 17 with a labeled ligand in the presence of test compounds, to form test reactions, and in the absence of test compounds, to form control reactions;

(b) incubating said test and control reactions under appropriate conditions to achieve equilibrium binding of said labeled ligand to hERβ;

(c) determining the level of binding of said labeled ligand to hERβ in said test and control cultures; and

(d) identifying as a hERβ-interactive compound any compound that reduces the binding of said labeled ligand to hERβ.

20. A method as defined in claim 19, wherein said ligand is 17-β estradiol.

21. A method as defined in claim 19, wherein said hERβ-interactive compound is an agonist.

22. A method as defined in claim 19, wherein said hERβ-interactive compound is an antagonist.

23. An antibody that specifically recognizes hERβ.

24. A method for identifying hERβ-interactive compounds, said method comprising:

(a) contacting the polypeptide of claim 18, which polypeptide encodes hERβ, with a labelled ligand in the presence of test compounds, to form test reactions, and in the absence of test compounds, to form control reactions;

(b) incubating said test and control reactions under appropriate conditions to achieve equilibrium binding of said labeled ligand to hERβ;

(c) determining the level of binding of said labeled ligand to hERβ in said test and control cultures; and

(d) identifying as a hERβ-interactive compound any compound that reduces the binding of said labeled ligand to hERβ.

25. A method as defined in claim 24, wherein said ligand is 17-β estradiol.

26. A method as defined in claim 24, wherein said hERβ-interactive compound is an agonist.

27. A method as defined in claim 24, wherein said hERβ-interactive compound is an antagonist.

28. The polypeptide of claim 17, wherein the polypeptide is modified with a label capable of providing a detectable signal.

29. The polypeptide of claim 28, wherein the signal is a radioisotope.

30. The polypeptide of claim 28, wherein the signal is a fluorescent compound.

31. The polypeptide of claim 18, wherein the polypeptide is modified with a label capable of providing a detectable signal.

32. The polypeptide of claim 31, wherein the signal is a radioisotope.

33. The polypeptide of claim 31, wherein the signal is a fluorescent compound.

34. The polypeptide of claim 17, wherein the polypeptide is produced in intact cells.

35. The polypeptide of claim 17, wherein the polypeptide is produced in cell-free translation systems.

36. The polypeptide of claim 18, wherein the polypeptide is produced in intact cells.

37. The polypeptide of claim 18, wherein the polypeptide is produced in cell-free translation systems.

38. The polypeptide of claim 17, wherein the polypeptide is chemically synthesized.

39. The polypeptide of claim 17, wherein the polypeptide is produced in a recombinant system.

40. The polypeptide of claim 18, wherein the polypeptide is chemically synthesized.

41. The polypeptide of claim 18, wherein the polypeptide is produced in a recombinant system.

42. (New) A purified polypeptide comprising amino acids 1-45 of the sequence depicted in Figure 4 SEQ ID NO:2, wherein when this polypeptide forms the N-terminus of human estrogen receptor β, the estrogen receptor β stimulates estrogen response element (ERE) activity to a greater extent than the truncated estrogen receptor lacking this N-terminal polypeptide sequence.

43. (New) A purified polypeptide comprising amino acids 1-45 of the sequence depicted in Figure 4 SEQ ID NO:2, wherein when this polypeptide forms the N-terminus of human estrogen receptor β, the estrogen receptor β attenuates NF-kB transcription activation while the truncated estrogen receptor lacking this N-terminal polypeptide sequence does not.

44. (New) A purified polypeptide comprising amino acids 1-45 of the sequence depicted in Figure 4 SEQ ID NO:2, wherein when this polypeptide forms the N-terminus of human estrogen receptor β, the estrogen receptor β is 2 to 3 times more active than the truncated estrogen receptor lacking this N-terminal polypeptide sequence in activating the ERE-reporter gene in the presence of estradiol.

45. (New) An isolated estrogen receptor-β comprising an N-terminus having amino acids 1-45 of the sequence depicted in Figure 4 SEQ ID NO:2, wherein the estrogen receptor β stimulates ERE activity to a greater extent than the truncated estrogen receptor lacking this N-terminal polypeptide sequence.

46. (New) An isolated estrogen receptor-β comprising an N-terminus having amino acids 1-45 of the sequence depicted in Figure 4 SEQ ID NO:2, wherein the estrogen receptor β attenuates NF-kB transcription activation while the truncated estrogen receptor lacking this N-terminal polypeptide sequence does not.

47. (New) An isolated estrogen receptor-β comprising an N-terminus having amino acids 1-45 of the sequence depicted in Figure 4 SEQ ID NO:2, wherein the estrogen receptor β is 2 to 3 times more active than the truncated estrogen receptor lacking this N-terminal polypeptide sequence in activating the ERE-reporter gene in the presence of estradiol.